

Nonlinear molecular excitations in a completely inhomogeneous DNA chain

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Abstract

We study the nonlinear dynamics of a completely inhomogeneous DNA chain which is governed by a perturbed sine-Gordon equation. A multiple scale perturbation analysis provides perturbed kink-antikink solitons to represent open state configuration with small fluctuation. The perturbation due to inhomogeneities changes the velocity of the soliton. However, the width of the soliton remains constant.

Key words:

DNA, Soliton, Multiple Scale Perturbation

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1 Introduction

A number of theoretical models have been proposed in the recent times to study the nonlinear dynamics of Deoxyribonucleic acid (DNA) molecule to understand the conservation and transformation of genetic information (see for e.g [1,2]). These models are based on longitudinal, transverse, rotational and stretching motions of bases. Among these different possible motions, rotational motion of bases is found to contribute more towards the opening of base pairs and to the nonlinear dynamics of DNA. The first contribution towards nonlinear dynamics of DNA was made by Englander and his co-workers [3] and they studied the base pair opening in DNA by taking into account the rotational motion. Yomosa [4,5] proposed a plane base rotator model by taking into account the rotational motion of bases in a plane normal to the helical axis, and Takeno and Homma generalized the same [6,7,8]. Later using this model, several authors found solitons to govern the fluctuation of DNA double helix between an open state and its equilibrium states [9,10,11,12,13,14]. Peyrard and Bishop [15,16] and Christiansen and his colleagues [17] studied the process of base pair opening by taking into account the transverse and longitudinal motions of bases in DNA. Very recently, there have been extensions of the radial model of Bishop and Peyrard [18,19], composite models for DNA torsion dynamics [20] and models interplaying between radial and torsional dynamics [21,22,23,24]. In all the above studies, homogeneous strands and hydrogen bonds have been considered for the analysis.

However, in nature the presence of different sites along the strands such as promoter, coding, terminator, etc., each of which has a specific sequence of bases is related to a particular function and thus making the strands site-dependent or inhomogeneous [25,26]. Also, the presence of abasic sites leads to inhomogeneity in stacking [27]. In this context, in a recent paper the present authors [28] studied the nonlinear molecular excitations in DNA with site-dependent stacking energy along the strands based on the plane base rotator model. The nonlinear dynamics of DNA in this case was found to be governed by a perturbed sine-Gordon (s-G) equation. The perturbed kink and antikink soliton solutions of the perturbed s-G equation represented an open state configuration of base pairs with small fluctuation. The perturbation in this case introduces small fluctuations in the localized region of the soliton retaining the overall shape of the soliton. However, the width of the soliton remains constant and the velocity changes for different inhomogeneities. The results indicate that the presence of inhomogeneity in stacking changes the number of base pairs that participate in the open state configuration and modifies the speed with which the open state configuration travels along the double helical chain. In reality, the presence of site-dependent strands in DNA changes the nature of hydrogen bonds between adjacent base pairs and the presence of abasic sites leads to absence of hydrogen bonds. Thus, when the strands are site-dependent in stacking, naturally the hydrogen bonds that connect the bases between the strands are also site-dependent. Hence, it has become

necessary to consider inhomogeneity in hydrogen bonds also in the study of nonlinear dynamics of DNA. In the present paper, we study the dynamics of DNA with inhomogeneity both in stacking and in hydrogen bonds using the plane base rotator model. The paper is organized as follows. In section 2, we present the Hamiltonian of our model and derive the associated dynamical equation for the inhomogeneous DNA. The effect of stacking and hydrogen bond inhomogeneity on base-pair opening is studied by solving the dynamical equations using a multiple scale soliton perturbation theory in section 3. The results are concluded in section 4.

2 Hamiltonian and the dynamical equation

We consider the B-form of a DNA double helix with site-dependent strands as well as base-pair sequence and study the nonlinear molecular excitations by considering a plane-base rotator model. In Fig. (1a) we have presented a sketch of the DNA double helix. Here, S and S' represent the two complementary strands in the DNA double helix and each arrow represents the direction of the base attached to the strand and the dots between arrows represent the net hydrogen bonding effect between the complementary bases. The z-axis is chosen along the helical axis of the DNA. Fig. (1b) represents a horizontal projection of the n^{th} base pair in the xy-plane. In this figure Q_n and Q'_n denote the tips of the n^{th} bases belonging to the strands S and S' . P_n and P'_n represent

the points where the bases in the n^{th} base pair are attached to the strands S and S' respectively.

As we are looking for opening of base pairs in DNA which is related to important DNA functions such as replication and transcription, we consider the rotational motion of bases (due to its importance among other motions) in a plane normal to the helical axis (z-direction) represented by the angles ϕ_n and ϕ'_n at the n^{th} site of the base pair. The stacking and hydrogen bonding energies are the major components of the energy in a DNA double helix. In the case of a homogeneous DNA system, Yomosa [4,5] expressed the Hamiltonian involving these energies in terms of the rotational angles ϕ_n and ϕ'_n under the plane base rotator model which was later modified by the present authors [28] in the case of site-dependent stacking. When both the stacking and hydrogen bonds are site-dependent, the Hamiltonian for our plane base rotator model of DNA double helix is written in terms of the rotational angles as

$$H = \sum_n \left[\frac{I}{2} (\dot{\phi}_n^2 + \dot{\phi}'_n{}^2) + J f_n \left[2 - \cos(\phi_{n+1} - \phi_n) - \cos(\phi'_{n+1} - \phi'_n) \right] - \eta g_n [1 - \cos(\phi_n - \phi'_n)] \right]. \quad (1)$$

The first two terms in the Hamiltonian (1) represent the kinetic energies of the rotational motion of the n^{th} nucleotide bases with I their moments of inertia and the remaining terms represent the potential energy due to stacking and hydrogen bonds. While J and η represent a measure of stack-

ing and hydrogen bonding energies respectively, f_n and g_n indicate the site-dependent (inhomogeneous) character of stacking and hydrogen bonds respectively.

The Hamilton's equations of motion corresponding to Hamiltonian (1) is written as

$$I \frac{\partial^2 \phi_n}{\partial t^2} = J [f_n \sin(\phi_{n+1} - \phi_n) - f_{n-1} \sin(\phi_n - \phi_{n-1})] + \eta g_n \sin(\phi_n - \phi'_n), \quad (2a)$$

$$I \frac{\partial^2 \phi'_n}{\partial t^2} = J [f_n \sin(\phi'_{n+1} - \phi'_n) - f_{n-1} \sin(\phi'_n - \phi'_{n-1})] + \eta g_n \sin(\phi'_n - \phi_n). \quad (2b)$$

It is expected that the difference in the angular rotation of neighbouring bases along the two strands in the case of B-form of DNA double helix is small [6,7].

Therefore under small angle approximation, in the continuum limit Eqs. (2a)

and (2b) are written as

$$\phi_{tt} = f(z) \phi_{zz} + f_z \phi_z - \frac{1}{2} g(z) \sin(\phi - \phi'), \quad (3a)$$

$$\phi'_{tt} = f(z) \phi'_{zz} + f_z \phi'_z - \frac{1}{2} g(z) \sin(\phi' - \phi). \quad (3b)$$

While writing Eqs. (3a) and (3b) we have chosen $\eta = -\frac{Ja^2}{2}$ and rescaled the time variable as $\hat{t} = \sqrt{\frac{Ja^2}{I}} t$. Now, adding and subtracting Eqs.(3a) and (3b) and by choosing $\phi = -\phi'$, we obtain

$$\Psi_{\hat{t}\hat{t}} - \Psi_{zz} + \sin \Psi = \epsilon [A(s(z)\Psi_z)_z - B h(z) \sin \Psi], \quad (4)$$

where $\Psi = 2\phi$. Further, as the contribution due to inhomogeneity is small compared to homogeneous stacking and hydrogen bonding energies while writing Eq.(4), the inhomogeneity in stacking and hydrogen bonding energies are expressed in terms of a small parameter ϵ as $f(z) = 1 + \epsilon As(z)$ and $g(z) = 1 + \epsilon Bh(z)$, where A and B are arbitrary constants. When $\epsilon = 0$, Eq. (4) reduces to the completely integrable sine-Gordon(s-G) equation which admits N-soliton solutions in the form of kink and antikink [29]. Hence, we call Eq. (4) as a perturbed sine-Gordon equation. For instance, the one soliton solution of the integrable s-G equation obtained through Inverse Scattering Transform (IST) method is written as,

$$\Psi(z, \hat{t}) = 4\arctan \exp[\pm m_0(z - v_0 \hat{t})], \quad m_0 = \frac{1}{\sqrt{1 - v_0^2}}. \quad (5)$$

Here v_0 and m_0^{-1} are real parameters that represent the velocity and width of the soliton respectively. In Eq.(5) while the upper sign corresponds to the kink soliton, the lower sign represents the antikink soliton. In Figs. (2a) and (2b) the one soliton kink-antikink solutions (Eq.(5)) are plotted by choosing $v_0 = 0.4$. The kink-antikink soliton solution of the integrable sine-Gordon equation describes an open state configuration in the DNA double helix. In Figure (2c), we present a sketch of how the base pairs open locally in the form of kink-antikink structure in each strand and propagate along the direction of the helical axis.

The base pair opening will help in the process of replication that duplicates DNA and in transcription which helps to synthesize messenger RNA. However, when $\epsilon \neq 0$, the inhomogeneity in stacking and hydrogen bonds may affect the base-pair opening through a perturbation on the kink-antikink solitons of the s-G equation. Therefore in the next section, we solve the perturbed s-G equation (4) using a multiple-scale soliton perturbation theory [30,31,32] (as has been carried out in the case corresponding to $B = 0$ [28]) to understand the effect of stacking and hydrogen bond inhomogeneities on the base pair opening.

3 Effect of stacking and hydrogen bonding inhomogeneities on the open state configuration

3.1 Multiple-scale soliton perturbation theory

In order to study the effect of inhomogeneity in stacking and hydrogen bonds on the base pair opening in the form of kink-antikink soliton by treating them as a perturbation, the time variable \hat{t} is transformed into several variables as $t_n = \epsilon^n \hat{t}$ where $n = 0, 1, 2, \dots$ and ϵ is a very small parameter [30,31,32]. In view of this, the time derivative and Ψ in Eq. (4) are replaced by the expansions $\frac{\partial}{\partial \hat{t}} = \frac{\partial}{\partial t_0} + \epsilon \frac{\partial}{\partial t_1} + \epsilon^2 \frac{\partial}{\partial t_2} + \dots$ and $\Psi = \Psi^{(0)} + \epsilon \Psi^{(1)} + \epsilon^2 \Psi^{(2)} + \dots$. We then equate the coefficients of different powers of ϵ and obtain the following equations. At $O(\epsilon^{(0)})$ we obtain the integrable sine-Gordon equation

$$\Psi_{t_0 t_0}^{(0)} - \Psi_{zz}^{(0)} + \sin \Psi^{(0)} = 0, \quad (6)$$

for which the one soliton solution takes the form as given in Eq.(5) with \hat{t} replaced by t_0 . Due to perturbation, the soliton parameters namely m and $\xi(\xi = v\hat{t})$ are now treated as functions of the slow time variables t_0, t_1, t_2, \dots . However, m is treated as independent of t_0 . The equation at $O(\epsilon^{(1)})$ takes the form

$$\Psi_{\tau\zeta}^{(1)} - \Psi_{\zeta\zeta}^{(1)} + (1 - 2\text{sech}^2\zeta)\Psi^{(1)} = F^{(1)}(\zeta, \tau), \quad (7)$$

where

$$\begin{aligned} F^{(1)}(\zeta, t_0) = & 2[As(\zeta)\text{sech}\zeta]_{\zeta} - 2aBh(\zeta)\tanh\zeta\text{sech}\zeta \\ & + 4v_0\text{sech}\zeta[m_{t_1} + (m^2\xi_{t_1} - \zeta m_{t_1})\tanh\zeta]. \end{aligned} \quad (8)$$

While writing the above equation we have used the transformation $\hat{\zeta} = m(z - vt_0)$ and $\hat{t}_0 = t_0$ to represent everything in a co-ordinate frame moving with the soliton. Then, we have used another set of transformations given by $\tau = \frac{\hat{t}_0}{2m} - \frac{(1+v)\hat{\zeta}}{2}$ and $\zeta = \hat{\zeta}$ for our later convenience. We have also replaced $\sin\Psi^{(0)}$ by $2a\tanh\zeta\text{sech}\zeta$, where $a = \pm 1$. The solution of Eq. (7) is searched by assuming $\Psi^{(1)}(\zeta, \tau) = X(\zeta)T(\tau)$ and $F^{(1)}(\zeta, \tau) = X_{\zeta}(\zeta)H(\tau)$. Substituting the above in Eq. (7) and simplifying we obtain

$$X_{\zeta\zeta} + (2\text{sech}^2\zeta - 1)X = \lambda_0 X_{\zeta}, \quad (9)$$

$$T_{\tau} - \lambda_0 T = H(\tau), \quad (10)$$

where λ_0 is a constant. Thus, the problem of constructing the perturbed soliton at this moment turns out to be solving Eqs. (9) and (10) by constructing the eigenfunctions and finding the eigen values [28,32].

It may be noted that Eq.(9) differs from the normal eigen value problem, with X_ζ in the right hand side instead of X . Hence, before actually solving the eigen value equation (9), we first consider it in a more general form given by

$$L_1 X = \lambda \tilde{X}, \quad L_1 = \partial_{\zeta\zeta} + 2\text{sech}^2\zeta - 1, \quad (11)$$

where λ is the eigen value. To find the adjoint eigen function to X , we consider another eigen value problem

$$L_2 \tilde{X} = \lambda X, \quad (12)$$

where L_2 is to be determined. Combining the two eigen value problems we get

$$L_2 L_1 X = \lambda^2 X, \quad L_1 L_2 \tilde{X} = \lambda^2 \tilde{X}. \quad (13)$$

From the above equations we conclude that the operator $L_1 L_2$ is the adjoint of $L_2 L_1$ and also X and \tilde{X} are expected to be adjoint eigen functions. Hence, we can find the eigenfunction by solving Eqs.(11) and (12). However, even-though L_1 is known as given in Eq.(11), the operator L_2 is still unknown. So, by experience we choose $L_2 = \partial_{\zeta\zeta} + 6\text{sech}^2\zeta - 1$, and solve the eigen value equations by choosing the eigen functions as

$$X(\zeta, k) = p(\zeta, k)e^{ik\zeta}, \quad \tilde{X}(\zeta, k) = q(\zeta, k)e^{ik\zeta}, \quad (14)$$

where k is the propagation constant. Further, as the operator L_2L_1 is self-adjoint, non-negative and satisfying a regular eigenvalue problem, the sine-Gordon soliton is expected to be stable. On substituting Eqs. (14) in Eqs.(11) and (12) in the asymptotic limit, we obtain the eigen value as $\lambda = -(1 + k^2)$. Now, in order to find the eigen functions we expand $p(\zeta, k)$ and $q(\zeta, k)$ [30,32] as

$$p(\zeta, k) = p_0 + p_1 \frac{\sinh \zeta}{\cosh \zeta} + p_2 \frac{1}{\cosh^2 \zeta} + p_3 \frac{\sinh \zeta}{\cosh^3 \zeta} + p_4 \frac{1}{\cosh^4 \zeta} + \dots, \quad (15a)$$

$$q(\zeta, k) = q_0 + q_1 \frac{\sinh \zeta}{\cosh \zeta} + q_2 \frac{1}{\cosh^2 \zeta} + q_3 \frac{\sinh \zeta}{\cosh^3 \zeta} + q_4 \frac{1}{\cosh^4 \zeta} + \dots, \quad (15b)$$

where p_j and q_j , $j=0,1,2,\dots$ are functions of k to be determined. On substituting Eqs. (14), (15a) and (15b) in Eqs. (11) and (12) and collecting the coefficients of 1, $\frac{\sinh \zeta}{\cosh \zeta}$, $\frac{1}{\cosh^2 \zeta}, \dots$ we get a set of simultaneous equations. On solving those equations we obtain the following eigen functions [28,32].

$$X(\zeta, k) = \frac{(1 - k^2 - 2ik \tanh \zeta)}{\sqrt{2\pi}(1 + k^2)} e^{ik\zeta}, \quad (16)$$

$$\tilde{X}(\zeta, k) = \frac{(1 - k^2 - 2ik \tanh \zeta - 2\text{sech}^2 \zeta)}{\sqrt{2\pi}(1 + k^2)} e^{ik\zeta}. \quad (17)$$

The higher order coefficients p_3, p_4, \dots and q_3, q_4, \dots vanish.

It may be noted that Eq.(10) is a linear inhomogeneous differential equation and can be solved using known procedures. The solution reads

$$T(\tau, k) = \frac{1}{i\lambda_0 k(1+k^2)} \int_{-\infty}^{\infty} d\zeta' F^{(1)}(\zeta', \tau) X^*(\zeta', k) (e^{\lambda_0[\tau - \frac{(1+v)}{2}\zeta']} - 1), \quad (18)$$

where $\lambda_0 = \frac{i(1+k^2)}{k}$. The first order correction to the soliton can be computed using the following expression.

$$\Psi^{(1)}(\zeta, \tau) = \int_{-\infty}^{\infty} X(\zeta, k) T(\tau, k) dk + \sum_{j=0,1} X_j(\zeta) T_j(\tau). \quad (19)$$

Here the continuous eigenfunctions $X(\zeta, k)$ and $T(\tau, k)$ are already known as given in Eqs. (16) and (18). However, the discrete eigen states X_0, X_1 and T_0, T_1 are unknown. The two discrete eigenstates X_0, X_1 corresponding to the discrete eigen value $\lambda = 0$ can be found using the completeness of the continuous eigenfunctions as

$$X_0(\zeta) = \text{sech}\zeta, \quad X_1(\zeta) = \zeta \text{sech}\zeta. \quad (20)$$

In order to find T_0 and T_1 , we substitute Eq.(19) in Eq.(7) and multiply by $X_0(\zeta)$ and $X_1(\zeta)$ separately and use the orthonormal relations to get

$$T_{1\tau}(\tau) = \int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau) X_0(\zeta) d\zeta, \quad (21a)$$

$$T_{0\tau}(\tau) - 2T_1(\tau) = - \int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau) X_1(\zeta) d\zeta. \quad (21b)$$

As $F^{(1)}(\zeta, \tau)$ given in Eq.(8) does not contain the time variable τ explicitly, the right hand side of Eqs. (21a) and (21b) also should be independent of

time, and hence we write the nonsecularity conditions as

$$\int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau) X_0(\zeta) d\zeta = 0, \quad (22a)$$

$$\int_{-\infty}^{\infty} F^{(1)}(\zeta, \tau) X_1(\zeta) d\zeta = 0. \quad (22b)$$

It may be indicated that, the nonsecularity conditions give way for the stability of soliton [33] under perturbation through an understanding of the evolution of soliton parameters such as width and velocity. Substituting the above equations in Eqs. (21a) and (21b), we choose $T_1(\tau) = 0$ and find $T_0(\tau) = C$ which has to be determined. For this, we substitute $T_1(\tau) = 0$ in Eq. (21b) and integrate to obtain

$$T_0(\tau) = \frac{(1+v)}{2} \int_{-\infty}^{\infty} d\zeta \zeta F^{(1)}(\zeta, \tau) X_1(\zeta). \quad (23)$$

In order to find the first order correction to soliton $\Psi^{(1)}$ we need to evaluate the eigen states $T(\tau, k)$ and $T_0(\tau)$ explicitly for which we have to find the explicit form of $F^{(1)}(\zeta, \tau)$ which contains unknown quantities like m_{t_1} , ξ_{t_1} , $s(\zeta)$ and $h(\zeta)$. Therefore, we substitute Eqs. (8) and (20) in the nonsecularity conditions given in Eqs. (22a) and (22b) and evaluate the integrals to obtain

$$m_{t_1} = -\frac{1}{2v_0} \int_{-\infty}^{\infty} \left(A [s(\zeta) \operatorname{sech} \zeta]_{\zeta} - a B h(\zeta) \operatorname{sech} \zeta \tanh \zeta \right) \operatorname{sech} \zeta d\zeta, \quad (24a)$$

$$\xi_{t_1} = -\frac{1}{2m^2 v_0} \int_{-\infty}^{\infty} \left(A [s(\zeta) \operatorname{sech} \zeta]_{\zeta} - a B h(\zeta) \operatorname{sech} \zeta \tanh \zeta \right) \zeta \operatorname{sech} \zeta d\zeta \quad (24b)$$

Here m_{t_1} and ξ_{t_1} represent the time variation of the inverse of the width and velocity of the soliton respectively.

3.2 *Variation of soliton parameters*

In order to find the variation of the width and velocity of the soliton while propagating along the inhomogeneous DNA chain, we assume that the soliton with a width of m_0^{-1} is travelling with a speed of $v_0(\xi_{t_0})$ when the perturbation is switched on. In other words, the problem now boils down to understand the propagation of the kink-antikink soliton in an inhomogeneous DNA chain and to measure the change of the soliton parameters due to inhomogeneity. To find the variation of the soliton parameters explicitly and to construct the perturbed soliton solution we have to evaluate the integrals found in the right hand sides of Eqs. (24a) and (24b) which can be carried out only on supplying specific forms of $s(\zeta)$ and $h(\zeta)$. Hence, we consider inhomogeneity in the form of localized and periodic functions separately and further assume that the inhomogeneity in the stacking and in the hydrogen bonds are equal. The localized inhomogeneity represents the intercalation of a compound between neighbouring base pairs or the presence of defect or the presence of abasic site in the DNA double helical chain. The periodic nature of inhomogeneity may represent a periodic repetition of similar base pairs along the helical chain.

3.2.1 Localized inhomogeneity

To understand the effect of localized inhomogeneity in stacking and hydrogen bonds on the width and velocity of the soliton during propagation, we substitute $s(\zeta) = h(\zeta) = \text{sech}\zeta$ in Eqs.(24a) and (24b). On evaluating the integrals, we obtain $m_{t_1} = 0$, $\xi_{t_1} = \frac{\pi}{12m^2v_0}(2A + aB)$ which can be written in terms of the original time variable \hat{t} by using the expansions $m_{\hat{t}} = m_{t_0} + \epsilon m_{t_1}$ and $\xi_{\hat{t}} = \xi_{t_0} + \epsilon \xi_{t_1}$ as

$$m = m_0, \quad \xi_{\hat{t}} \equiv v = v_0 + \frac{\epsilon\pi(2A + aB)}{12m^2v_0}, \quad (25)$$

where $1/m_0$ is the width and v_0 is the velocity of the soliton in the absence of inhomogeneity. The first of Eq.(25) says that when the inhomogeneities in stacking and in hydrogen bonds are in the form $s(\zeta) = h(\zeta) = \text{sech}\zeta$, the width of the soliton remains constant, thereby showing that the number of base pairs participating in the opening process remain constant during propagation. However, from the second of Eq.(25), we find that the velocity of the soliton gets a correction. The nature of correction in the velocity depends on the value of ‘ a ’ which takes $+1$ or -1 and also on the nature of A and B which can be either positive or negative. When A and B are greater than zero, the inhomogeneity will correspond to an energetic barrier and on the other hand when A and B are less than zero, the inhomogeneity will correspond to a potential well. First, we consider the case corresponding to $a = +1$. In this case

when $(2A + B) > 0$, the velocity of the soliton gets a positive correction and it may propagate along the chain without formation of a bound state. On the other hand, when $(2A + B) < 0$, the inhomogeneities slow down the soliton. Ofcourse, when $(2A + B) = 0$, that is when the inhomogeneities in the stacking and in the hydrogen bonding suitably balance each other, the velocity of the soliton remains unaltered. Finally, the soliton stops when the original velocity satisfies the condition $v_0^2 = \frac{1}{1 - \frac{12}{\epsilon\pi(2A+B)}}$. In all the above cases, the stability of the soliton is guaranteed. A similar argument can be made in the case when $a = -1$ with $(2A + B)$ replaced by $(2A - B)$. It may also be noted that similar results have been obtained in the case of resonant kink-impurity interaction and kink scattering in a perturbed sine-Gordon model by Zhang Fei et al [34], say that the kink will pass the impurity and escape to the positive infinity when the initial velocity of kink soliton is larger than the critical value. In a similar context Yakushevich et al [35] while studying the interaction between soliton and the point defect in DNA chain showed numerically, that the solitons are stable. At this point it is also worth mentioning that Dandoloﬀ and Saxena [36] realized that in the case of an XY-coupled spin chain model which is identifiable with our plane-base rotator model of DNA, the ansatz $sech\zeta$ energetically favours the deformation of spin chain.

3.2.2 Periodic inhomogeneity

We then choose the periodic inhomogeneity in the form $s(\zeta) = h(\zeta) = \cos \zeta$ and substitute the same in Eqs. (24a) and (24b). On evaluating the integrals, we get $m_{t_1} = 0$, $\xi_{t_1} = \frac{\pi[\pi A + a(4-\pi)B]}{16m^2v_0}$ which can be written in terms of the original time variable \hat{t} as

$$m = m_0, \quad \xi_{\hat{t}} \equiv v = v_0 + \frac{\epsilon\pi[\pi A + a(4-\pi)B]}{16m^2v_0}. \quad (26)$$

From Eq.(26), we observe that the width of the soliton remains constant and the velocity gets a correction. Here also, one can project an argument similar to the case of localized inhomogeneity. The only difference between the two cases is the quantum of correction added to soliton velocity. Normally in DNAs, inhomogeneity in hydrogen bonds is expected to be dominant and therefore one would expect that $B > A$. One can verify that it is possible to obtain the above condition from the velocity corrections in Eqs. (25) and (26) by writing $\frac{\epsilon\pi[\pi A + (4-\pi)B]}{16m^2v_0} > \frac{\epsilon\pi(2A+B)}{12m^2v_0}$ when $a = +1$. The above condition indicates that the correction in velocity in the case of periodic inhomogeneity is larger than that in the case of localized inhomogeneity. This is because in this case, the inhomogeneity occurs periodically in the entire length of the DNA chain. In a recent paper, Yakushevich et al [35] studied numerically the dynamics of topological solitons describing open states in an inhomogeneous DNA and investigated interaction of soliton with the inhomogeneity and the results, have

very close analogy with the results of our perturbation analysis. It was shown that the soliton can easily propagate along the DNA chain without forming a bound state thus showing that soliton moving with sufficiently large velocity along the DNA chain is stable with respect to defect or inhomogeneity.

3.3 First order perturbed soliton

Having understood the variation of the width and velocity of the soliton in a slow time scale due to perturbation, we now construct the first order perturbed soliton by substituting the values of the basis functions $\{X\} \equiv \{X(\zeta, k), X_0(\zeta), X_1(\zeta)\}$ and $\{T\} \equiv \{T(\tau, k), T_0(\tau), T_1(\tau)\}$ given in Eqs. (16), (20), (18), (23) with $T_1(\tau) = 0$ in Eq.(19), we get

$$\begin{aligned}
\Psi^{(1)}(\zeta, t_0) = & -\frac{1}{\pi} \int_{-\infty}^{\infty} dk \frac{1}{(1+k^2)^3} (1-k^2-2ik \tanh \zeta) e^{ik\zeta} \\
& \times \int_{-\infty}^{\infty} d\zeta' \left([As(\zeta') sech \zeta']_{\zeta'} - aBh(\zeta') sech \zeta' \tanh \zeta' + 2v_0 sech \zeta' \right. \\
& \times \left[m_{t_1} + (m^2 \xi_{t_1} - \zeta' m_{t_1}) \tanh \zeta' \right] \left(1-k^2+2ik \tanh \zeta' \right) e^{-ik\zeta'} \\
& \times \left(e^{\lambda_0[\tau + \frac{(1+v_0)}{2}\zeta']} - 1 \right) + (1+v) sech \zeta \int_{-\infty}^{\infty} d\zeta' \left([As(\zeta') sech \zeta']_{\zeta'} \right. \\
& - aBh(\zeta') sech \zeta' \tanh \zeta' + 2v_0 sech \zeta' [m_{t_1} \\
& \left. + (m^2 \xi_{t_1} - \zeta' m_{t_1}) \tanh \zeta'] \right) \zeta'^2 sech \zeta'. \tag{27}
\end{aligned}$$

While writing the above, we have also used $\tau = \frac{1}{2m}[t_0 - m(1+v)\zeta]$. It may be noted that majority of the poles that lie within the contour in Eq. (27), are purely imaginary giving rise to exponentially localized residues and hence do not give rise to any radiation thus will lead to stable form of soliton [37].

3.3.1 Localized inhomogeneity

Now, we explicitly construct the first order perturbation correction to the one soliton in the case of the localized inhomogeneity (i.e) when $s(\zeta) = h(\zeta) = \text{sech}\zeta$, by substituting the corresponding values of $F^{(1)}$, m_{t_1} and ξ_{t_1} in Eq. (27). The resultant integrals are then evaluated using standard residue theorem [38] which involves very lengthy algebra and at the end we obtain

$$\begin{aligned} \Psi^{(1)}(\zeta, t_0) \approx (2A + aB) & \left[\frac{40}{27\sqrt{2}} \sqrt{\text{sech}\zeta} e^{-\frac{3\zeta}{2}} + \frac{32}{27\sqrt[3]{2}} \tanh \zeta \sqrt[3]{\text{sech}\zeta} e^{-\frac{5}{3}\zeta} \right. \\ & \left. + \frac{\pi}{12v^2} [2vt_0 + m^2(v^2 - 1)] \text{sech}\zeta \right]. \end{aligned} \quad (28)$$

Finally, the perturbed one soliton solution, that is $\Psi(z, t_0) = \Psi^{(0)}(z, t_0) + \Psi^{(1)}(z, t_0)$ (choosing $\epsilon = 1$) is written in terms of the original variables as

$$\begin{aligned} \Psi(z, t_0) \approx 4 \arctan \exp[\pm m_0(z - v_0 t_0)] & + (2A + aB) \left[\frac{40}{27\sqrt{2}} e^{\mp \frac{3(m(z - vt_0))}{2}} \right. \\ & \times \sqrt{\text{sech}[\pm m(z - vt_0)]} + \frac{32}{27\sqrt[3]{2}} \tanh[\pm m(z - vt_0)] \\ & \times \sqrt[3]{\text{sech}[\pm m(z - vt_0)]} e^{\mp \frac{5}{3}m(z - vt_0)} + \frac{\pi}{12mv^2} \\ & \left. \times [m(v^2 - 1) + 2t_0 v] \text{sech}[\pm m(z - vt_0)] \right]. \end{aligned} \quad (29)$$

Knowing Ψ , the angle of rotation of bases $\phi(z, t_0)$ can be immediately written down by using the relation $\phi = \frac{\Psi}{2}$. In Figs. 3(a,b) we have plotted $\phi(z, t_0)$, the rotation of bases under the perturbation $g(z) = h(z) = \text{sech}z$ by choosing $a = A = B = 1$ and $v_0 = 0.4$. From the figures we observe that there appears

fluctuation in the form of a train of pulses closely resembling the shape of the inhomogeneity profile in the width of the soliton as time progresses. But, there is no change in the topological character and no fluctuations appear in the asymptotic region of the soliton. In Figs. (4a) and (4b), we have plotted the perturbed kink and antikink solitons respectively corresponding to the case when the hydrogen bond inhomogeneity is absent ($B=0$) for comparison. From Figs. (3) and (4), we observe that the fluctuation in the form of a train of pulses appear in both the cases. Eventhough the perturbed solitons in both the cases appear qualitatively the same, the inhomogeneity in hydrogen bonds adds more fluctuation in the width of the soliton.

3.3.2 Periodic inhomogeneity

We then repeat the procedure for constructing the perturbed one soliton solution $\Psi(\zeta, t_0)$ in the case of periodic inhomogeneity by choosing $s(\zeta) = h(\zeta) = \cos \zeta$, and obtain the perturbed soliton solution as

$$\Psi(z, t_0) \approx 4 \arctan \exp[\pm m_0(z - v_0 t_0)] + [A\pi + aB(4 - \pi)][b + dt_0] \operatorname{sech}[m_0(z - vt_0)], \quad (30)$$

where $b = \frac{\pi^2}{16v^2}(v^2 - 1)$ and $d = \frac{\pi^2}{8m_0v}$. In Figs. 5(a,b), we plot the angle of rotation of bases $\phi(= \frac{\Psi}{2})$, using the perturbed soliton given in Eq.(30) for the same parametric choices as in the case of localized inhomogeneity. We observe from the figures that, the fluctuation appears in the width of

the soliton without any change in its topological character asymptotically. In order to compare the results with that of the case when the hydrogen bonding inhomogeneity is absent as also found in [28](see Figs. (6a,b)), we plot the perturbed soliton found in (30) when $B = 0$. As in the previous case, here also we observe that fluctuation appears in the width of the soliton in both the cases and the inhomogeneity in hydrogen bonds introduces more fluctuation.

4 Conclusions

In this paper, we studied the nonlinear dynamics of a completely inhomogeneous (inhomogeneity in both stacking and hydrogen bonding) DNA double helix by considering the dynamical plane-base rotator model. The dynamics of this model in the continuum limit gives rise to a perturbed sine-Gordon equation, which was derived from the Hamiltonian consisting of site-dependent stacking and hydrogen bonding energies. In the unperturbed limit, the dynamics is governed by the kink-antikink soliton of the integrable sine-Gordon equation which represents the opening of base pairs in a homogeneous DNA. In order to understand the effect of inhomogeneity in stacking and hydrogen bonds on the base pair opening, we carried out a perturbation analysis using multiple-scale soliton perturbation theory. The perturbation not only modifies the shape of the soliton but also introduces change in the velocity of the soliton. From the results, we observe that when the inhomogeneity is in a lo-

calized or periodic form, the width of the soliton remains constant. However, the velocity of the soliton increases, decreases or remains uniform and even the soliton stops, depending on the values of inhomogeneity represented by A and B . The soliton in all the cases are found to be stable. From the results of the perturbed soliton we observe that the inhomogeneity in stacking and hydrogen bonds in both the cases (localized and periodic forms) introduce fluctuation in the form of pulses in the width of the soliton. However, there is no change in the topological character of the soliton in the asymptotic region (see Figs. 3 and 5). The fluctuation is more when both the inhomogeneities (site-dependent stacking and hydrogen bonds) are present, whereas it is less in the case of homogeneous hydrogen bonds and site-dependent stacking. Hence, we conclude that, the addition of inhomogeneity in hydrogen bonds does not introduce big changes in the soliton parameters and shape except a correction in the velocity of the soliton and fluctuation. The above dynamical behaviour may act as energetic activators of the RNA-polymerase transport process during transcription in DNA. In the case of short DNA chains the discreteness effect assumes importance and hence we will analyse the discrete dynamical equations (2) and the results will be published elsewhere.

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Fig.1. (a) A schematic structure of the B-form DNA double helix. (b) A horizontal projection of the n^{th} base pair in the xy-plane.

Fig.2. (a) Kink and (b) antikink soliton solutions of the sine-Gordon equation (Eq.(4) when $\epsilon = 0$) with $v_0 = 0.4$. (c) A sketch of the formation of open state configuration in terms of kink-antikink soliton in DNA double helix.

Fig.3.(a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = h(z) = \text{sech}z$ with $A = B = 1$ and $v_0 = 0.4$.

Fig.4. (a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $s(z) = \text{sech}z, h(z) = 0$ with $A = 1$ and $v_0 = 0.4$.

Fig.5.(a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = h(z) = \cos z$ with $A = B = 1$ and $v_0 = 0.4$.

Fig.6.(a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = \cos z, h(z) = 0$ with $A = 1$ and $v_0 = 0.4$.

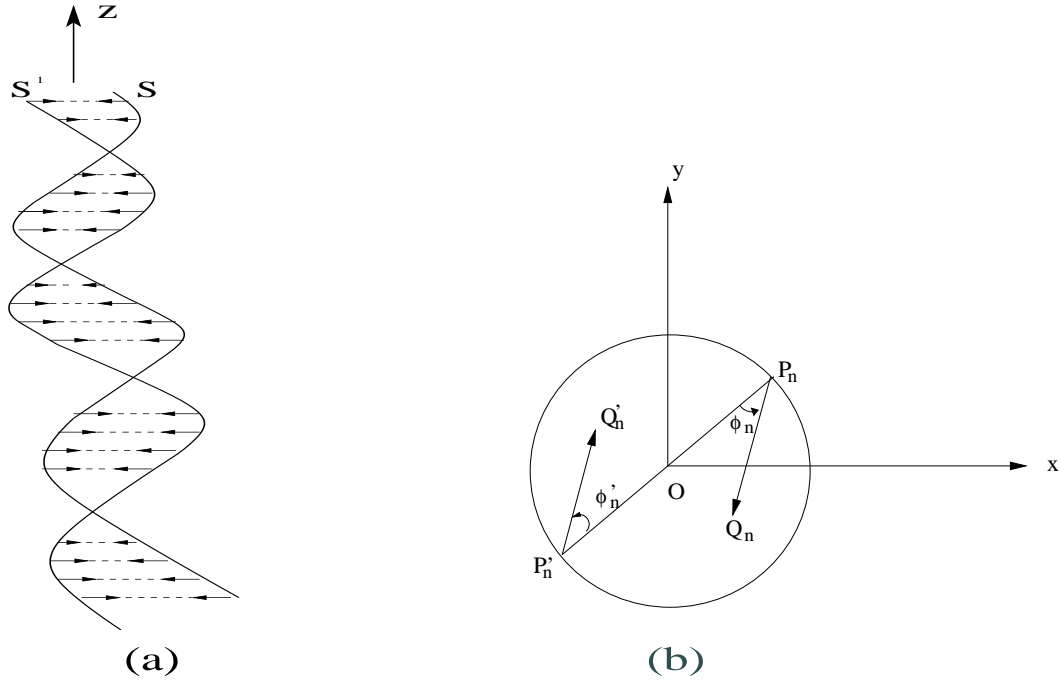


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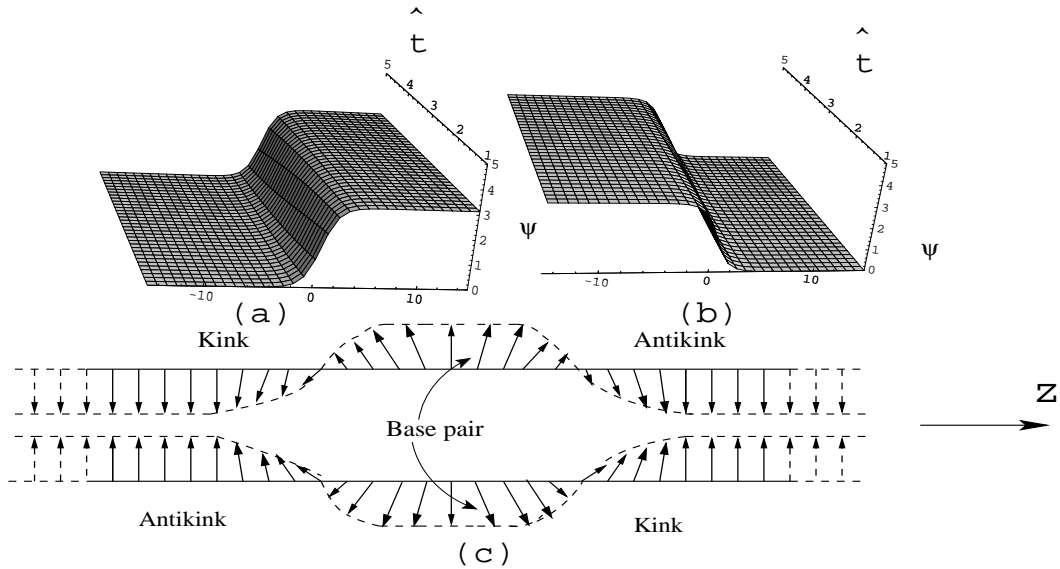


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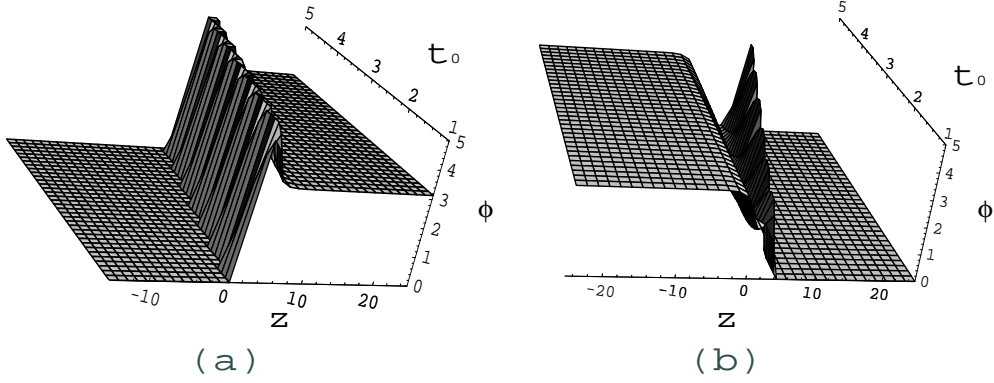


Fig. 3. (a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = h(z) = \text{sech } z$ with $A = B = 1$ and $v_0 = 0.4$.

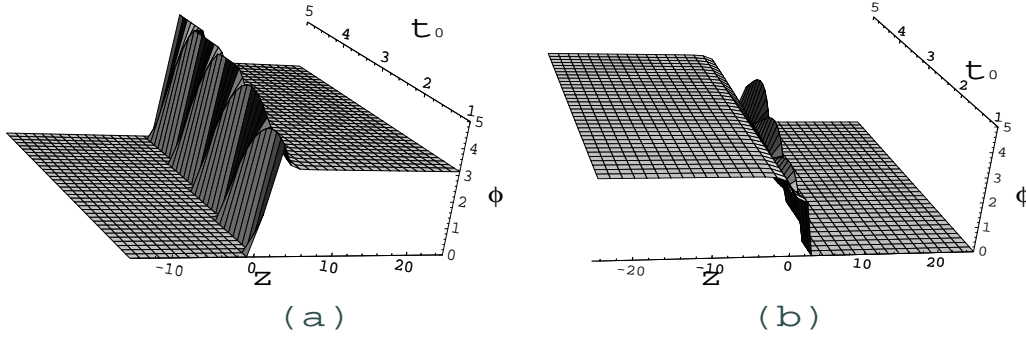


Fig. 4. (a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $s(z) = \text{sech } z, h(z) = 0$ with $A = 1$ and $v_0 = 0.4$.

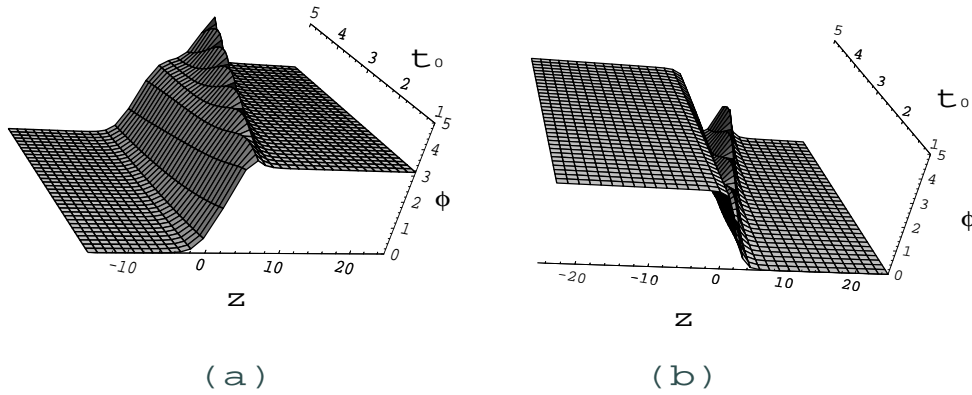


Fig. 5. (a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = h(z) = \cos z$ with $A = B = 1$ and $v_0 = 0.4$.

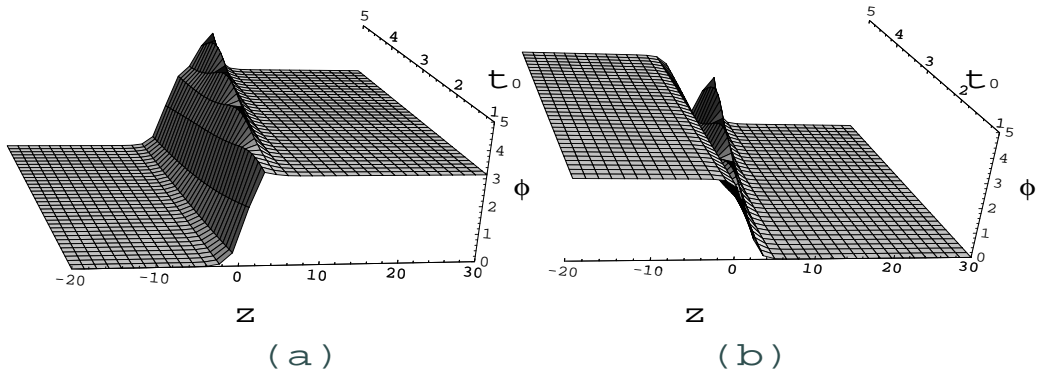


Fig. 6. (a) The perturbed kink-soliton and (b) the perturbed antikink-soliton for the inhomogeneity $g(z) = \cos z, h(z) = 0$ with $A = 1$ and $v_0 = 0.4$.